

The Obstetrician's Responsibility in Infant Mortality

T. FLOYD BELL, M.D., Oakland

SUMMARY

Although infant mortality has been remarkably reduced, stillbirth and neonatal death rates have been improved very little. Efforts at lowering the fetal death rate must be directed to those conditions affecting the fetus during labor or immediately afterward.

Prevention of premature labor and better care of the premature infant during labor and the neonatal period offer hope of a greater salvage of premature infants. Proper environment and trained personnel are necessary.

Spontaneous delivery is safest for the infant. Difficult operative procedures are associated with a high incidence of birth trauma, asphyxia and death.

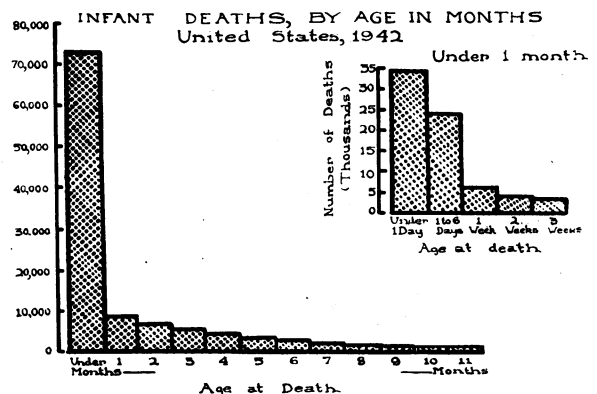
Since asphyxia is one of the chief causes of infant death, prevention, diagnosis and treatment of it are most important. Administration of oxygen to mildly asphyxiated infants before the injury has become irreversible may help to prevent late manifestations of anoxia.

DURING the past decade, statistics from the United States Bureau of the Census have shown considerable improvement in maternal and infant mortality, although less as to infants than mothers. Between 1933 and 1943 the maternal mortality declined 60 per cent.⁴ During the same period deaths of infants (under 1 year of age) decreased from 58.1 per 1,000 live births, to 40.4, a decrease of 31 per cent. This was further reduced to 38.3 in 1945¹³—quite a contrast to a rate of 99.9 in 1915. This gratifying picture can be further improved.

The reduction in infant mortality has not been equal in all age groups. This is shown in the accompanying graph of statistics for the year 1942. The rates of decline vary inversely with the age, the greatest number of deaths occurring during the first month of life, and most of these in the first day.

In 1943, 60 per cent of the infant deaths were due to prenatal or natal causes; in 1944, 61 per cent. Neonatal mortality was regarded as perhaps the most important subject discussed at the recent International Congress in Dublin, indicating the importance of fetal mortality.¹⁴

Presented as Chairman's Address before the Section on Obstetrics and Gynecology at the 77th Annual Session of the California Medical Association, San Francisco, April 11-14, 1948.



Prematurity is by far the leading cause of infant mortality.⁴ In 1944, 30 per cent of infant deaths in the United States were ascribed to premature births; in 1943, 29 per cent. Premature birth, congenital malformations, birth trauma and congenital debility are the more outstanding prenatal and natal causes accounting for 54 per cent of all infant deaths in 1944 and 53 per cent in 1943. Although there is considerable difference in infant mortality among races, that feature will not enter into this discussion.

To improve the present mortality rate our efforts and attention must first be directed to saving those infants lost during the prenatal and neonatal period. Prenatal care, management of labor and delivery, and initiation of respiration immediately after birth are all important factors in life for the full term as well as for the premature infant. The responsibility for this early care must be assumed by the obstetrician. Beck¹ in his study of infant mortality has emphasized the obstetrician's responsibility for the hazards of the first few days of life.

In premature infants the vital organs are insufficiently developed to carry on the functions necessary to maintain life. Increased efforts must be made to give prematures careful and intelligent attention after delivery. The best environment possible must be supplied under adequate supervision by specially trained personnel.

Increased efforts must be made to prevent premature delivery. Mauzey⁷ reports the probable causes of prematurity were chiefly eclamptogenic toxemia, premature detachment of the placenta, syphilis, multiple pregnancy, habitual abortion, placenta previa, nephritic toxemia, heart disease and pyelitis. More complete prenatal care and institution of proper treatment offer hope of greater fetal salvage in these as well as other causes of premature delivery. The induction of labor and cesarean sec-

tion for nonmedical reasons, except in definitely mature infants, should be discouraged. Whenever feasible, maternal complications of the prenatal period, which are associated with a high incidence of premature labor, should be treated conservatively as long as the delay does not create a serious hazard to the mother.

Since the premature infant is more susceptible to injury than the full term infant, oxytocic substances should be avoided in order to prevent rapid and precipitate labor. The membranes should be preserved until full dilatation of the cervix occurs. Heavy sedation is to be avoided in premature labor. Immediately after delivery the respiratory passages must be cleared by aspiration and energetic efforts made to start the infant breathing at once. The most common causes of death during the neonatal period, among both premature and full term infants, are pulmonary lesions such as atelectasis, massive aspiration and pneumonia. While some cells from the amniotic fluid are to be found in the respiratory passages without significance, aspiration of large amounts of amniotic fluid may occur before delivery and produce very distinct symptoms. Labate⁵ found in his series that aspiration of foodstuff was a common cause of pneumonia. This is a preventable cause of death and can be greatly reduced by proper care and properly trained attendants.

Birth injury stands high as a cause of fetal death. The premature infant is especially liable to injury. Because of the possibility of trauma, delivery by high forceps and other difficult means carries a heavy risk for the infant. Even in cesarean section there is risk of fatal injury of the infant. The lowest rate of infant loss is in those delivered spontaneously. Deaths due to birth injury are reported to have accounted for 9.2 per cent of all deaths of infants in the United States for 1944. D'Esopo and Marchetti² in their study ascribed 11.5 per cent to this cause, while Potter and Adair⁸ reported 13 per cent. Rapid expulsive contractions with a short labor predispose to trauma, particularly in prematures, even though the actual delivery be spontaneous.

PROLONGED LABOR INCREASES DEATHS

It is generally known that the infant death rate is directly proportional to the length of labor. D'Esopo and Marchetti² found that in labors over 30 hours the fetal death rate was doubled. In any prolonged labor consultation is advisable unless delivery seems close at hand.

Asphyxia, or anoxia, is often given as the cause of death. Hypoxemia¹¹ and transnatal asphyxia and anoxia⁶ are other terms that have been used to describe this condition. Anoxia may be related to a number of factors such as birth trauma, cord complications, premature separation of the placenta, heavy sedation and anesthesia. D'Esopo and Marchetti² found in nearly 20 per cent of their cases that infant deaths were ascribed to asphyxia; Potter and Adair⁸ reported 28 per cent. Many of such deaths occur before or during delivery, resulting in

stillbirths. The deaths are rarely due to diseases which are intrinsic in the fetus. Interference with intra-uterine circulation caused by prolapse of the cord or tight cord about the neck of the fetus is responsible for asphyxia in many cases. Intrapartum bleeding from premature detachment of the placenta, or from placenta previa, may interfere sufficiently with the oxygen exchange to produce death from asphyxia. A less acute condition such as the so-called infarcted placenta may cut down the nourishment as well as the oxygen supply to the fetus, resulting in a poorly nourished anoxic infant which may be lost immediately or not survive the neonatal period.

In cases of anoxia the unwise use of anesthetics or sedatives may produce additional risk to the already distressed infant. These agents have little effect until after delivery, when they act as respiratory depressants, especially in the premature.

EFFECT OF ANOXIA ON CHILD DEVELOPMENT

Even though the immediate danger to the infant from anoxia may have passed, the more remote and late effects of such a state of oxygen want may produce serious handicaps to the child. Many authors have brought this to attention. The experimental work of Windle¹² on animal fetuses subjected to low oxygen tension is most convincing. His motion pictures of such animals showed poorly coordinated movements and symptoms of damage to the higher nerve centers. Autopsy disclosed varying degrees of damage to the central nervous system, depending on the degree of anoxia. Several workers have demonstrated what are probably the late effects of low oxygen tensions on children suffering from difficult psychological problems. Preston⁹ studied the effect of anoxia on the subsequent behavior of children. More than one-fourth were of subnormal intelligence. Arrest of physical, mental, nervous, emotional and personality development occurred throughout the series. Faber has also emphasized this point. Schreiber¹⁰ had already shown the relationship between asphyxia and mental disease. These changes represent permanent damage to the central nervous cells as a result of oxygen want. A deficiency of oxygen for more than a few minutes results in irreparable damage to the nerve cells.

The diagnosis of asphyxia in the newborn offers no problem after delivery. Before delivery, a fetal pulse rate 30 or 40 beats slower than the previous basic rate is evidence of real fetal distress. This is the only reliable sign of impending danger to the fetus. Attempts at forcible delivery at this time only add to the danger of trauma and more anoxia. Administration of oxygen to the mother is an effective means of supplying oxygen to the asphyxiated fetus. In the individual case, if no improvement is noticed after a few minutes, one may assume this method is of no value.

Many observers have emphasized the importance in caring for the newborn in a state of asphyxia.

The older methods of slapping, swinging, hot and cold tubbing, are obsolete and have no place in modern methods of resuscitation. The first essential is an open and free respiratory tract. Oxygen must reach the alveoli of the infant's lungs. Even in partial or mild degrees of asphyxia, the administration of oxygen will prevent permanent damage to the nerve cells, unless the damage is sufficient to be irreversible. It is important to supply oxygen until the child has good color, breathes well and seems normal. Body heat must be maintained, especially in prematures.

Even though the pediatrician ultimately takes care of the newborn, proper nutrition and hygiene of the mother during the prenatal period, expert management of labor and delivery, and the institution of well established respiration are responsibilities of the obstetrician. He must deliver the infant to the pediatrician in the best condition possible.

431 Thirtieth Street.

REFERENCES

1. Beck, A. C.: Obstetrician's responsibility for the hazards of the first few days of life with special reference to anoxia and prematurity, *Am. J. O. & G.*, 51:173-183 (Feb.), 1946.
2. D'Esopo, D. A., and Marchetti, A. A.: Causes of fetal and neonatal mortality, *Am. J. O. & G.*, 44:1-22 (July), 1942.
3. Dunham, E. C.: Deaths of premature infants in the United States, U. S. Children's Bureau Stat., Series No. 2.
4. Gooch, M.: Ten years of progress in reducing maternal and infant mortality, *Child*, 10:77-83 (Nov.), 1945.
5. Labate, J. S.: A study of the causes of fetal and neonatal mortality on the obstetric service of Bellevue Hospital, *Am. Jr. O. & G.*, 54:188-199, 1947.
6. Lund, C. J.: Transnatal asphyxia and anoxia, *West. J. Surg.*, 50:575-578 (Nov.), 1942.
7. Mauzey, A. J.: Obstetric factors in premature birth, *Am. J. O. & G.*, 39:295-301 (Feb.), 1940.
8. Potter, E. L., and Adair, F. L.: Clinical-pathologic study of the infant and fetal mortality for a ten-year period at the Chicago Lying-In Hospital, *Am. J. O. & G.*, 45:1054-1065 (June), 1943.
9. Preston, M. I.: Late behavioral aspects found in cases of prenatal, natal and postnatal anoxia, *J. Pediat.*, 26:353-366 (April), 1945.
10. Schreiber, F.: Mental deficiency from prenatal asphyxia, *Proc. Am. A. Ment. Deficiency*, 63:95-106, 1939.
11. Traut, H. F.: Hypoxemia of the fetus, *West. J. Surg.*, 54:379-383 (Oct.), 1946.
12. Windle, W. F., and Becker, R. F.: Asphyxia neonatorum, *Am. J. O. & G.*, 45:183-199 (Feb.), 1943.
13. Infant mortality, *J.A.M.A.*, 136:492 (Feb. 14), 1948.
14. The International Congress, Rotunda Bicentenary, *Lancet*, 2:105, 1947.

